

## b.—PATHOLOGY OF THE NERVOUS SYSTEM AND MIND AND PATHOLOGICAL ANATOMY.

**APHASIA.**—In the *Gaz. des Hôpitaux*, No. 132, 1876, M. Luys gives an account of a woman aged 40, who died at the Salpêtrière of slow asphyxia dependent on a bulbar lesion by descending atrophy of the pyramidal bundles. Eight years previously she had suffered with a complete right hemiplegia, the remote primary cause of which was traced back to acute articular rheumatism affecting the endocardium. With the hemiplegic attack, there was complete aphasia, which, according to the patient's account lasted about eighteen months, and then speech was very rapidly recovered. At the time of admission the speech was good; intelligence apparently intact, and the motor power to a considerable extent regained.

At the autopsy, some very striking changes were presented in the two sides of the brain; on the left side there was a spot of softening involving especially the third frontal convolution, the posterior portion of which was destroyed. The convolutions of the insula, the inferior point of conjunction of the frontal and ascending parietal, had disappeared, the first temporal was also involved, in fact, the cortical substance of the whole region of the insula and its marginal convolutions was abolished. The ascending parietal and frontal convolutions were intact in their upper three-fourths as likewise was the angular gyrus of the second and third temporal. On the inner face of the hemisphere, the first frontal, the paracentral lobe, and the crested convolution were intact; the quadrilateral lobe, on the contrary, was less developed than that of the opposite side. The grey substance of the nucleus lenticularis was completely destroyed, that of the nucleus caudatus persisted in the shape of a remnant; the optic thalamus was notably atrophied.

The right hemisphere on the other hand, presented a remarkable development in comparison with the other. This difference may be relatively represented by the following data of the weight of the two after their immersion in a bath of nitric acid; previous to drying, the left hemisphere weighed two hundred and forty-five grammes (7 2.3 oz.), the right weighed three hundred and sixty grammes (11 1/4 oz.), a difference of one hundred and fifteen grammes (3 7.12 oz.).

In this hemisphere there was noted a remarkable development of the first, the second, and especially of the third frontal convolutions. The ascending frontal and ascending parietal were likewise notably developed. The superior parietal region joining the ascending parietal had acquired a volume and sinuities quite unusual in this region. The angular gyrus and the first temporal convolution were of the normal dimensions; the second and third temporal on the other hand were hard to define.

On its internal face the same hemisphere, passing from front to rear, showed a very marked development of the first frontal convolution which was very flexuous, also the paracentral lobule was strongly developed. The crested convolution was likewise clearly marked, and the quadrilateral lobe also; the fusiform convolution was very notably developed. The convolutions of the insula were very clearly defined.

The central nuclei also partook of the augmentation of volume observed in the hemisphere generally.

The left cerebral peduncle was very notably atrophied, and this atrophy extended across the pons; the pyramid of the opposite side was also affected. The same was the case with the most internal portion of the left anterior pyramid. Some lacunae existed in the gray substance of the pons.

In reply to a question of M. Ranvier in the Soc. de Biologie, when the paper was read, M. Luys stated that no microscopic examination was made. Even without this, however, the communication is of great interest and suggestiveness.

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**HYSTERIA.**—Dr. Frank Woodbury, in an interesting paper entitled "On Cases called Hysterical," in the *Med. and Surg. Reporter*, Dec. 2, finishes with the following conclusions:

1. What has been called Hysteria is not a disease, but a symptom of disease.

2. Where the pathological source of such symptom resides in the uterus or ovaries, cases may, with some show of propriety, be termed hysterical.

3. Where the uterus and organs associated with it in function are not in a morbid condition, no symptoms can be correctly called hysterical.

4. When the diagnosis of hysteria is made, the burden of proof rests with the user of the term, to show, first, that there is co-existing uterine disorder, and secondly, that it is the direct and sole cause of the pathological phenomena in question.

5. Symptoms called hysterical may be due to reflex irritation of the great nerve centres.

6. The source of the irritation causing the reflex symptoms may reside in any other organ than the uterus.

7. Where the cause exists elsewhere than with the uterus, the symptoms are improperly termed hysterical.

8. As there is nothing in the symptoms themselves to indicate whether they are uterine, or not uterine, in their origin, the word Hysteria is of doubtful propriety, being in one case incorrectly applied, and in the other having nothing to commend it, that would counterbalance its positive disadvantage of imposing a definite pathological character upon a disease in advance of the diagnosis.

9. Medical nomenclature offers more precise expressions for the various uterine diseases than the word hysteria, while its use to describe a pure neurosis is evidently incorrect. In all cases called hysterical, the diagnostician should not be misled by a name given to a group of symptoms,

but should investigate their nature and source, and apply, in preference, a title that more clearly describes their pathological relations.

10. The progress of pathology requires the use of the word Hysteria should be very much restricted, if not finally discontinued.

SECONDARY DEGENERATIONS OF THE CORD FROM CORTICAL CEREBRAL LESIONS.—At the Soc. de Biologie, Oct. 21, (rep. in *Gaz. des Hôpitaux*, No. 124, 1876), M. Pitres offered a communication relative to the secondary degenerations of the cord in cases of cortical lesions of the brain. These secondary degenerations are not observed in all cases of cortical lesions. Certain ones, at present perfectly well known, can produce them. It results, in effect, from a great mass of observations collected by M. Pitres in the service of M. Charcot:

1. That every lesion of the brain, of whatever extent, if without the cortical motor zone, cannot produce these secondary descending degenerations of the cord.

2. That a lesion, even of very slight extent, occupying the motor points of the cerebral cortex, causes this descending secondary degeneration.

M. Pitres exhibited a number of histological specimens in support of these views.

M. Charcot called attention to the importance of these facts in relation to the question of cerebral localizations. They are indeed physical facts anatomically demonstrating the existence of certain localizations. Thus if the ascending and descending parietal convolutions and the paracentral lobe are injured, no matter to how slight an extent, it gives rise to secondary degenerations of the spinal cord, to a gross lesion visible even to the unaided eye, consisting of a gray sclerous band which may extend even as far as the lower portions of the cord. All the other regions of the brain may be previously destroyed without causing this secondary degeneration; it is not produced so long as the regions we have named are respected. These regions have not therefore the same anatomical connections as the others; they seem to form a brain within a brain.

PATHOLOGICAL ANATOMY OF THE FACIAL PARALYSIS OF INFANTS CAUSED BY THE USE OF THE FORCEPS.—J. Parrot, and E. Troisier, *Archives de Tocologie* Aug. 1876 (abstr. in *La France Médicale*).

Paralysis in the face in new born infants, caused by the use of the forceps, was first noticed about the year, 1837 by P. Dubois, and has since been well described in the thesis of Landouzy. Its pathological anatomy, however, still remains to be determined. In all the autopsies hitherto published, the facial nerve seemed either intact or very slightly injured. The following are the results of these observations by the authors.

Alterations of the facial nerve exist, especially a diminution of consistency and change of color; the nerve appears either grayish red or grayish and semi-transparent when separated from its neurilemma. At the horizon of the stylo-mastoid foramen, we find a well marked line of demarca-

tion between the external portion which presents the appearance described and the cranial portion which preserves its normal characters. Microscopic examination shows that the nerve is steatose for its whole extent. The myeline of all the tubes is replaced by fatty matter which, according to its stage of degeneration, appears in larger or smaller globules or very minute granulations. We find no trace of the cylinder axis, and the nuclei of the neurilemma and the perineurium are fewer than in the normal condition. These alterations are met with in the same degree from the stylo-mastoid foramen to the muscular filaments in the muscles of the face.

In one of these observations in which the examination was made twelve days after birth, the muscles presented no appreciable alteration, but in the other two cases, in which the trouble was a month old, the muscles of the paralyzed side, were found to be less in volume and less ruddy in color than those of the opposite side. The fibres, for the most part, preserved their transverse striae, and did not seem to have been lessened in size, but it is probable that a certain number of them were atrophied and that others had even disappeared entirely.

We have, therefore, the degeneration of the nerve and simple atrophy of the muscles innervated by it; these lesions evidently resulting from pressure exerted upon the nerve even to the stylo-mastoid foramen. The paralysis makes itself evident immediately after the application of the forceps, as the contusion produced by the instrument is sufficiently severe and prolonged to destroy the relation between the museles and the nervous centres. It persists as long as the consecutive degeneration of the nerve exists, and disappears as soon as the regeneration takes place. The average duration of the affection, which always terminates favorably, is six weeks. If the paralysis lasts only some hours or some days, there is reason to think that the contusion of the nerve has not been sufficient to alter its nutrition, and that it therefore only temporarily destroys its functions, and we can understand also, why the paralysis affects sometimes only a part (upper or lower) of the face, if the compression was limited to particular branches of the facial nerve.

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TRAUMATIC LESIONS OF THE SYMPATHETIC.—Dr. A. Seeligmüller offered to the surgical section of the *Deutsche Naturforscher Versammlung* at its meeting last year in Hamburg, the following short paper which we extract from the *Deutsche med. Wochenschrift*, of Oct. 28.

Gentlemen: If I permit myself to bring before you a subject of not special surgical interest, you will excuse me, since surgeons are the ones who are really in the position to best observe traumatic lesions of the cervical sympathetic—affections that are by no means as rare as would appear from the small number of reported cases. In support of this statement, I can say that of the thirteen cases published, up to the present date, I myself, with quite limited opportunities of observation, have seen eight, plainly, because during the past six years, I have given special attention to the subject. Of these thirteen cases symptoms of paralysis were observed in ten, of irritation in only three.

As in physiological experiments, so the symptoms of traumatic lesions of the cervical sympathetic may be divided in three series: *viz.*, oculo-pupillary, vaso-motor, and trophic phenomena.

1. As oculo-pupillary symptoms in paralysis of the cervical sympathetic, we observe, partial closure of the eye, contraction of the pupil, and enophthalmus; in cases of irritation of the sympathetic, there was widening of the lid-opening, prominence of the orbit, and dilatation of the pupil. It would be going too far, should I dwell upon these symptoms. The most constant are the pupillary ones; the advance or retrocession of the orbit cannot always be made out with certainty. In regard to the width of the space between the eyelids, I have observed in two cases of irritation of the sympathetic, that it was not, as was expected, dilated, but notably contracted. Especially do the at present received views of the functions of the Mueller's muscle fibres need revision, since there are still other phenomena not in agreement with them. For example, we ascribe to the smooth muscular fibres in the eyelids the function of opening their eye. How comes it that in paralysis of the motor oculi, the eyelids remain shut in spite of them?

2. The second class of phenomena, the vaso-motor are much more rarely observed, than the oculo-pupillary ones. In many cases I think I have found the explanation of this peculiarity, in that the vaso-motor phenomena are not continuous, but appear spasmodically and in definite "vaso-motor attacks." These vascular symptoms in ten cases of paralysis of the sympathetic were observed only twice; in one, there is only a greater redness of the side of the face corresponding to the lesion, and in the other I observe besides a heightened temperature. In the three cases of irritative lesion of the sympathetic, described by myself, in only one did I detect vaso-motor phenomena, but in this, in so perfect a degree, that even a non-professional eye would at once notice the contrast between the pallor on the side of the lesion and the rosy whiskey-blossom tint of the other side of the face. Moreover the temperature in the meatus of the injured side was almost a degree centigrade lower than that of the other.

3. Trophic disorders are also comparatively rare, only five times in thirteen cases. As regards the space of time after the injury in which these should show themselves, we lack sufficient data. Still it seems that they may appear with extraordinary rapidity in cases of irritation of the sympathetic. In the one case, in which the vaso-motor symptoms were so prominent, the wasting of the cheeks was so far advanced on the affected side, in eight days time that not only I observed it, but the patient himself noticed it without having his attention directed thereto. In these cases of atrophy after irritation of the sympathetic, we can most readily explain the nutrition disturbances following the injury so rapidly, with Bruuner, by a chronic contraction of the vessels. In cases of paralysis of the sympathetic, on the other hand, we can find no satisfactory explanation for the complicating hemiatrophy, in my opinion, except under the condition of admitting special trophic nerves.

Finally, as regards the nature and locality of the lesion in the ten cases of paralysis; in six, it was a gunshot wound, in one a punctured wound,

twice, fracture of the clavicle, and once a severe contusion in the region of the shoulder, and in the three cases of irritative trouble, the cause was contusion of the shoulder, once with, and twice without fracture of the collar-bone.

As is well known, Hutchinson has called attention to the frequent coincidence of paralysis of the brachial plexus with affections of the cervical sympathetic. That this statement of Hutchinson's is not altogether incorrect, is shown by the fact that of our thirteen cases, in nine was the brachial plexus either partially or wholly paralyzed. In such cases it is probable that, not the main cord of the sympathetic but the rami communicantes between it and the brachial plexus affords the parts, wounding of which produces the sympathetic symptoms.

In regard to this point the experiments I have undertaken on animals have produced as yet no certain results. A greater amount of clinical material would be useful in determining more satisfactorily the special regions, injury of which has its consequence, the usual sympathetic phenomena, and would allow us to draw conclusions as to the points of selection for the lesion that will involve more or less exclusively the fibres of the sympathetic.

It would afford me great pleasure if this short communication should induce closer observation for complications on the side of the sympathetic in all cases of injury in the region of the neck and shoulder, for I am assured that if that were done, the number of observed cases of traumatic lesions of the cervical sympathetic would soon become comparatively large.

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**THE DISORDERS OF SENSIBILITY IN IDIOPATHIC CONTRACTURES OF THE EXTREMITIES.**—At the session of the medical section of the French Association for the Advancement of Science last summer (rep. in *Gaz. des Hôpitaux*, Sept. 12), M. Manouvriez fils (of Valenciennes), drew from the histories of six observations of idiopathic contracture of the extremities, the following conclusions.

1. Besides various sensory troubles (painful or simply abnormal sensations), there constantly exists in tetany, outside of the attacks and after their definite cessation, a more or less accentuated paralysis of the sensibility to pain, to contact, to temperature and to tickling, of the skin, and often even of the mucous membranes; a paralysis having its locality preferably in the parts affected by the contractures and accompanied only exceptionally with alterations of sense. These alterations of sensibility, accompanied, moreover, with circulatory disturbances, justify the employment of bromide of potassium, which has been tried with good results by the author against this disease.

2. In the study of sensory paralyses, the aesthesiometer is indispensable to rigorously appreciate the state of tactile sensibility.

3. Clinically the analgesia resolves itself into analgesia properly so-called, or loss of sensibility to pain, so to speak, physiological, immediate or provoked, and into anodynia, or abolition of the sensibility to pain, so to speak, pathological, consecutive or spontaneous. (In one case, in

fact, a burn which was not felt at once as an injury, was felt later as a pathological process.)

4. The various kinds of sensibility (æsthesia, algæsia, odynia, pallesthesia, thærmæsthesia, the muscular sense, and even the sense of taste, may be altered independently of each other. Perhaps it may some day be possible to show that special conductors correspond to these various sensibilities or at least that the peripheral terminal corpuscles for each of them are anatomically distinct.

ALTERATIONS OF THE BRAIN AND CARDIAC GANGLIA IN HYDROPHOBIA. —Wassilieff, *Centralbl. f. d. med. Wissensch.* No. 36, Sept. 2, gives the results of microscopic examination of the brain and heart of a young woman dead from hydrophobia. The parts of the brain examined were the hemispheres, the corpora striata, the thalami optici, the pons, the medulla and the cerebellum.

The microscopic examination of the hardened and colored sections gave the following results:

1. Certain nerve cells of the medulla appeared muddy, their contours dimmed, and their nuclei obscured. Similar but more marked appearances were observed in certain of the cerebellar Purkinjes cells.
2. In the interstitial tissue of the brain there were noticed a great number of indifferent round elements, of the size of white blood corpuscles, which were very strongly tinged by the coloring matter employed. These elements (probably due to exudated white blood corpuscles) were most numerous in or in the vicinity of the perivascular spaces, though in some cases they were seen grouped six or ten together, at a distance from them in the neuroglia (proliferated neuroglia nuclei?). Finally such occur in the pericellular spaces and even in the protoplasm of the nerve cells (Kolesnikoff).
3. The blood vessels were strongly dilated and filled with blood corpuscles, the endothelium swollen in stellate patches; here and there were vessels of which the walls consisted of a finely granular, yellowish, strongly refracting substance soluble neither in absolute alcohol nor in turpentine. The most striking phenomenon, however, was the presence of the special, slightly shining, strongly refracting substance in the perivascular spaces, especially in the cortex of the hemispheres. Sometimes this substance so collected around the vessel, that in cross section it appeared to be surrounded with an irregular ring, which exercised so strong a pressure upon it that it was perceptibly narrowed; in other cases this hyaloid (according to Benedikt) substance laid in little masses, which frequently surrounded the vessel so regularly that it suggested epithelium. This hyaloid substance would take no coloring substance, and was soluble neither in strong alkalies (boiling with caustic potash), nor in powerful acids (acetic and hydrochloric); the same negative result was met with, with the employment of alcohol, turpentine, and the reaction on the amyloid substance. In other parts of the brain the perivascular spaces were more or less dilated.

In the ganglia of the heart the following appearances were noted.

1. The endothelium of the sheath surrounding the nerve cells was

swollen in stellate patches; and within the sheath and in the interstitial tissue of the ganglion were round elements of the size of a white blood corpuscle. The blood vessels around the ganglion, the great vein stem only excepted, seemed for the most part free from blood. 2. In the nerve cell itself, the protoplasm had more or less of a muddy appearance and on this account the nuclei were much obscured or quite invisible; in some cells there could be seen a collection of a finely granulated pigmentary substance. The most remarkable and unfailing peculiarity was that the nerve cells did not perfectly fill the sheath of the ganglion, but that there existed between them a free space through which the processes of the cells pass. (An altogether similar appearance was noted by Lubimoff in the cervical sympathetic ganglia in œdema from heart disease.)

In order to ascertain with certainty whether this appearance was due to œdema or to shrinkage of the cells, the author made very careful micrometer measurements and for comparison shows similar ones made by Dr. Iwanoff on normal ganglia. The result proved that the phenomenon observed was not due to shrinkage of the cells, but to expansion of the sheaths due to œdema.

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THE INFLUENCE OF POLITICAL AND SOCIAL COMMOTIONS ON THE DEVELOPMENT OF MENTAL DISEASES.—The following are the conclusions of a recent work by M. Lunier, as given in the *Gaz. des Hôpitaux*, No. 108, 1876.

The events of 1870–1871 caused, more or less directly, between the 1st of July, 1870 to the 31st of December, the explosion of seventeen or eighteen hundred cases of insanity. During this same period the French asylums received thirteen hundred less patients than in the corresponding period of 1869 and '70. The events of 1870–'71, therefore, had the immediate result of considerably diminishing the number of admissions into the asylums, and consequently the number of those remaining at the end of the year. The number of insane patients on the first of January, 1872, which ought to have been, all things being equal, 40,056 was in reality only 37,451, a difference of 2,605 below what might under normal conditions have been expected. The diminution of the number of admissions between July 1st, 1870 and Dec. 11, 1871, should be attributed to various direct and indirect causes, among which it is necessary to cite: *a.* the disturbance produced in the service of the asylums by the events of these years; *b.* the parsimony of certain departmental administrations; *c.* the suspension of certain etiological influences, which in periods of calm and prosperity often produce mental alienation.

The character as to the acuteness of the cases of insanity observed in 1870–'71, and their consequent rapid termination by death, or far more frequently by recovery, has likewise contributed in a certain degree to diminish the number of the patients remaining at the end of these years. But at the close of the year 1871, the number of admissions began to increase, in and 1872, it presented an altogether exceptional increase (2,785); in 1873 the additions were only 872, a proportion very near the average. This *recrudescence* in the number of admissions, which, moreover, must be at



tributed to diverse causes, and the character of chronicity, and hence the hopeless nature of the disease in many of the newly admitted, had the effect of augmenting in very large proportion; starting with the year 1872, the number of those remaining at the end of the year, which was 40,236 at the end of 1872, and 41,108 at the end of 1873. According to all the probabilities, this last figure differs very little from that which would have been the case, without the occurrence of the disastrous years we are considering.

The occurrences of 1870-'71 temporarily diminished, but did not arrest the progressive increase of the relative number of insane placed in special asylums, which was in 1869 about 1 in 989 inhabitants, and in 1874, 1 in 964. The increase in insanity since the beginning of 1872 has been, moreover, nearly uniform throughout France.

The mental diseases caused by the occurrences of 1870-'71 were more frequent among males than among females. The increase in the entries since 1872, seems, on the other hand, to affect females more than males, but in both cases the difference is slight. Hereditary predisposition played only a relatively unimportant part in the genesis of mental alienation due to the events of these years. It has only been noted in 24 cases in 100, while under ordinary conditions its influence in various degrees, is seen in 63 cases in 100.

Among the cases of mental disease attributed to the occurrences of 1870-'71, some act only indirectly by provoking emotions that are often in ordinary times causes of insanity; but which during these years were more numerous and more accentuated; others acted directly on the individual. These last have been observed only in the departments occupied or threatened by the enemy; the others on the contrary, have been observed in all parts of France. The most frequently observed determining causes were, the inquietude produced by the enemy's approach, the shame and chagrin at being under their flags, the departure for the army of a cherished friend, the physical and moral fatigues of the war,—and especially the siege of Paris,—the emotions felt during a battle or a bombardment, changes of position or fortune resulting from the events, chagrin caused by the news of our reverses, politico-social excitements, the occupation of the country by the enemy. Although the causes of insanity in the patients were especially of a depressing and debilitating kind, we have observed nearly every one of the forms and varieties of mental disease commonly met with in the asylums. The expansive forms were even more frequent than the depressive ones.

If, therefore, the perturbations produced by physical causes in the functions of the brain present almost constantly the same characters, those induced by moral causes have generally no relations or only accidental relations, with the causes that bring them about. An attentive study of cases of relapse shows that in the same individual; A, the same moral cause may induce altogether diverse forms of insanity; B, causes completely unlike, produce sometimes the same and sometimes different forms of insanity.

In many of our patients, nevertheless, notably those of intemperate habits, or who were profoundly anæmic, certain symptoms of the disease suggested up to a certain point, the causes that had induced it. Among

the more frequently observed morbid phenomena, we ought to mention stupor, panophobia anxiety, sitiophobia, ideas of suicide, megalomania, hallucinations of hearing, and insane ideas of persecution.

**CERTAIN REFLEX SYMPTOMS IN NERVOUS DISEASES.**—We copy the following from a recent article on certain reflex symptoms in nervous diseases by Dr. V. Henze from the *St. Petersburger med. Wochenschr.* No. 35, Oct. 30, 1876.

The reflex symptoms on which I may say a few words are the following:

1. The *sinew reflex* of Erb and Westphal (*Arch. f. Psych.* 1875, V. p. 792-802 and p. 803-835). Erb found that if when in a sitting position, the lower leg dependent and the foot unsupported, the ligamentum patella below the knee or the quadriceps tendon above it is lightly percussed, the leg undergoes a quick involuntary extension. Erb called this, already well known phenomenon, the "knee phenomenon." Further, repeated, passive dorsal flexions of the foot produce rapidly repeated contractions of the muscles of the calf (*foot phenomenon* of Erb). Westphal (l. c.) confirmed Erb's statements; both found that in certain affections of the cord (tabes) these reflexes were lacking, but that they were exalted in others, (lateral sclerosis).

2. The *cremaster reflex* of Jastrowitz (*Berl. klin. Wochenschr.* No. 31, 1875) serves as a diagnostic sign in dubious cases of unconsciousness in consequence of cerebral apoplexy and similar causes, in which one cannot determine the paralyzed side, and is produced by strong pressure on the thigh of the side examined, a hand-breadth above the internal condyle of the femur. If there is no paralysis, the testicle of the corresponding side is drawn up by contraction of the cremaster, but this does not occur on the paralyzed side, (Dr. Henze states in a note, that he and his colleagues have often tried this test in recent cases of apoplexy, but in most cases without decided results; often mere raising and dropping the limb is sufficient to decide as to its paralysis, etc.).

3. The *abdominal muscular reflex* of Rosenbach (*Arch. f. Psych.* VI, 1876, p. 845). Rosenbach, repeating Jastrowitz' experiments, sought for a similar symptom that would not be dependent on sex, and found it in the contraction, or failure to contract, of the abdominal muscles when the finger nail is passed over the surface or cold substances are applied. He found in ten cases of cerebral hemiplegia that the cremaster and abdominal reflexes were lacking on the paralyzed side, the sinew reflex was, on the other hand, usually exalted. Tests as to the relation of smooth muscular fibres to cerebral hemiplegias on the nipples, showed that they did not contract or wrinkle on the paralyzed side from tickling. I shall name this fourth reflex the *nipple reflex*.

I have in my service alone, (in the nervous department of the Obukhow Hospital) examined these reflexes in sixteen hemiplegias and two tabetic patients, on three suffering from spinal meningitis, two from spinal myelitis and three from sciatica, twenty-six in all, and will here report the results. I would mention here that the cremaster and abdominal reflexes

are very easily excited by light faradization of the skin over the places named in healthy persons on the unparalyzed side of the hemiplegias. I found in hemiplegias, adding the ten cases of Rosenbach, the following:

The sinew reflex was present in the paralyzed side of fourteen cases out of seventeen; the cremaster reflex was wanting on the paralyzed side eighteen times in twenty-one cases, the abdominal reflex twenty-one times in twenty-five, the nipple reflex eighteen times in twenty-two cases. The disagreements of these members among themselves is due to the fact that Rosenbach did not observe all the reflexes. In one case of right hemiplegia complicated with aphasia, of five years duration when observed, the cremaster reflex was very pronounced on the paralyzed side while all the others failed; the motor paralysis of the arm and leg was well toward recovery, the sensible paralysis diminished, but the aphasia persisted. There were, it may be mentioned, decided ataxic movements of the right hand.

The statements of Rosenbach seem to me to be sufficiently confirmed. I must still add that the contractions of the abdominal muscles and the nipples cannot always be well distinguished, and that I made the most of my observations in a rather dark summer barrack, where many insignificant contractions may have escaped me. I cannot agree with Eulenberg's opinion that these muscular reflexes more commonly fail in recent cases, for I could not satisfy myself that the duration of the disease had such an influence.

The statement of Westphal that the sinew reflex fails in ataxia is confirmed by my two cases, it also failed in the cases of myelitis. Those of meningitis gave no constant result. In the three cases of sciatica it was interesting to notice that the cremaster reflex was suppressed during the disease and reappeared again upon recovery.

The question now occurs how these reflexes take place. The first author, Erb, describing the sinew reflex, laid the "knee phenomenon" directly to mechanical irritation of the sinew of the patella and quadriceps. The "foot phenomenon" is explained by him as follows: that by the tension of the tendo-Achillis a contraction of the calf muscles is incited in a reflex way; by cessation of this contraction a spasm causing extension again takes place, and soon. Westphal thinks that the cause of each contraction must be sought in a direct mechanical phenomenon,—extension of the muscle through concussion of the tendon, and although the existence of sensory nerves in the tendons is doubtful, their presence is not an essential condition of these reflexes. These very plausible theories, which have strong support in the well known great mechanical irritability of the muscles, especially in pathological conditions, are contradicted by some very valuable experiments on animals by Schultz and Feurbringer (*Centralbl. f. d. med. Wissensch.* No. 54, 1875). They experimented upon rabbits, with the following results. 1. After laying bare the patellar sinew and the surrounding muscles they caused contraction of the quadriceps and the flexors of the foot by a blow on the tendon, not only of the same but of the opposite side. 2. Division of the quadriceps tendon did not prevent the phenomenon. 3. Section of the crural nerve destroyed the sinew reflex, but not the mechanical irritability of the muscles (in

opposition to Westphal's views). 4. Paralysis of the nerve by curare destroyed the sinew reflex but left the mechanical irritability of the muscles intact. These experiments clearly showed that for the sinew reflex, as for other reflexes, the integrity of the sensory nerves was a *sine qua non*. But the presence of such nerves in the sinews of a number of animals has been proved by Sachs (*Reichert a DuBois Reymond's Archiv.* IV. 1875). Sachs says (*Deutsch med. Wochenschr.* No. 5, 1876): The point of entrance and course of the nerves show characteristic relation for each separate sinew. The final termination, as a rule, is in the passage into its non-medullated branchlets which are perceived thick and bushy in a plate-like mass. In an interesting sinew (M. Sterno radialis, Cuvier) there are peculiar terminal bodies (*Schneuenendkolben*) plainly for the perception of the tension which the sinew receives from the muscular force.

The route of the sinew reflex is somewhat as follows; sensory nerves of the sinew, crural nerve, the four upper lumbar nerves as sensory routes, the lumbar cord as the place for the reflex centre, and the motor fibres of the crural, the muscular nerves of the quadriceps and the extensors of the foot as the motor routes.

As to the suppression of the cremaster reflex in the paralyzed, Jastrowitz thinks that it depends directly upon a motor disorder of the abdominal musculature, set up simultaneously with the other motor disturbances by the apoplectic lesion. Since the cremaster is formed from the muscular bundles of only the lower portion of the united mass of the transverse and oblique muscles, and moves with them, he assumes a paralysis also of this part. To the possible objection to this view, that so partial a paralysis of two muscles could not take place leaving so much of them intact, he offers the fact of the not very infrequent paralysis of the gluteal muscles in hemiplegia.

The routes of the cremaster reflex are; nervous cutaneous femor. ant. med., the two sapheni, and the anterior branch of the obturator as sensory routes, the upper part of the lumbar cord as relay, and the external spermatic nerve as a motor route.

Rosenbach also refers the failure of the abdominal muscle reflex to a paralysis of the abdominal muscles, and rightly denies the possibility of a direct muscular irritation acting in the production of these reflexes, as when we strike the muscles with a percussion hammer.

As routes for this reflex, he gives the ilio-hypogastric and ilio-inguinal nerves, and the anterior abdominal cutaneous nerves as the sensory ones. the lumbar and dorsal cord as reflex centres, and the ilio-hypogastric nerve as the route of motor transmission.

The nipple reflex is only incidentally here of interest.

In reviewing the experiments for the explanation of the absence of these reflexes in hemiplegias, it appears that no observer has paid attention to the almost inevitable accompaniment of the motor paralysis, the hemianæsthesia. I very often found in my examinations the points from which these reflexes are excited, anæsthetic, and this fact suggests to me the question whether or not the paralysis of the sensible integument or the sinew nerves, was not the cause of the failure of the phenomenon, and not the paralysis of the muscle. The cause of the sensory paralysis in hemiplegia, as well as in

the spinal affections is in the nervous centres, which, in case the peripheral irritation is not conducted to it by sensory nerves, cannot reflect the impulse along the motor nerves. The above mentioned case in which with the recovery of the central apparatus the reflex also re-appeared.

My material is still too scanty, and the time as yet too short to produce all the desired results; but it seemed to me that I ought to assert my view, since, should it prove correct, it would afford a very useful symptom for the prognosis of hemiplegias, in the re-appearance of these reflexes. Clinically, these reflexes have no very great importance, except the sinew reflex. I desire with my communication to incite investigation in other interesting points in regard to this subject. Perhaps it would be in looking up the following questions.

1. What are the relations to each other as to time of the motor and sensory paralysis, both in regard to their appearance and their disappearance?
2. Are the muscular reflexes always absent in unilateral anæsthesia?
3. Is the improvement in the motor paralysis parallel with the re-appearance of the reflexes?

SPASMODIC TABES DORSALIS.—Isidore Bétous *These de Paris*, 1876 (Abstr. in *Rev. des. Sci. Med.*)

Under this name the author describes a rare affection of the nervous system, already noticed by Erb, and to which M. Charcot has devoted an interesting lecture. The dominant character of this new disease is spasm and contracture, at first slight, but which become more and more pronounced and soon reduces the patient to complete debility. Motility is alone affected in these patients, the reverse being the case with ataxies in whom various troubles exist. It is rational to suppose that the anatomical lesions occupy the lateral columns, but the lack of an autopsy up to the present time prevents any localization of the alteration in the cord.

The evolution of spasmodic tabes dorsalis is eminently slow; it may be divided into three periods characterized each by special symptoms:

1. Gradual paresis of the inferior limbs, accompanied rarely with painful symptoms; no disorders of sensibility.
2. Contracture invading the same lower limbs and affecting the progressive in a peculiar manner. Spontaneous and induced tremors, the former constant.
3. The paresis and contracture attack the superior members; this condition may persist, improve or disappear. The contracture of the inferior members, increases so far that progression generally becomes impossible; it sometimes affects the abdominal muscles.

Sensibility is always preserved intact, spontaneous painful sensations are rare. There is neither muscular atrophy nor rectal, vesical, or genital disorders. The general state of health is good, the cerebral functions are accomplished normally.

This affection should not be confounded with transverse myelitis. The latter begins brusquely, is accompanied with paraplegia, urinary disorders, anæsthesia, and only in the late stages of the disorder do we see contracture

and tremors. It is the same with myelitis by compression and locomotor ataxia, which differ essentially from spasmodic tabes. Multiple sclerosis resembles this disease somewhat in paraplegia of the lower limbs, and the tremor, but we never meet in this affliction with the tremor of the members, the embarrassment of speech and the disorders of intelligence, and of the centres which are the regular thing in multiple sclerosis.

Amiotrophic lateral sclerosis, is of all spinal affections, the one that most resembles spasmodic tabes, but the muscular atrophy of the superior members, never met with in tabetis, suffices to distinguish it.

The progress of the disease is slow, and it does not endanger life.

Treatment has so far been ineffectual, still the use of the ascending current, and hydropathy are to be advised.

TWO DIFFERENT FORMS OF TETANUS DIAGNOSED BY PNEUMOGRAPHY.  
Ch. Richet *Gaz. Méd. de Paris*, No. 14, 1876, (Abstr. in *Rev. des Sci. Méd.*)

Death in tetanus coming on most frequently from asphyxia due to contraction of the respiratory muscles, it becomes a matter of interest to make a minute analysis of the mechanical phenomena of the respiration by means of the pneumograph.

The respiratory trace obtained some hours before death from a patient suffering from tetanus showed that at certain moments there was a sort of pause in the respiration and that it occurred during the expiration. There was therefore expiratory spasm, one that might involve the expiratory muscles and those of the glottis.

In another case death occurred in a sort of ill-characterized comatose condition, but without asphyxia. But in him the tetanic pause was during inspiration and not expiration.

Thus, in those two cases we have marked differences, since in the first there was arrest of expiration and in the second, arrest of inspiration. One practical consequence follows. If the spasm is due to the inspiratory muscles, tracheotomy is useless, and we may hope for a cure since the hæmatosis will suffice to permit therapeutic agents being absorbed and modifying the medullary excitation. If, on the other hand, there is spasm of expiration the prognosis will be graver, since hæmatosis at a given moment will be *null* or insufficient, and asphyxia imminent. In such cases as these M. Verneuil has been able to revive by tracheotomy the unfortunate victims of tetanus who were becoming asphyxiated.

Thus in a diagnostic point of view in view of the prognosis and the treatment, it is of importance to recognize two forms of tetanus: that in which the tetanic spasm affects inspiration, and the other, in which it involves the expiration.

ALBUMINURIA AS A RESULT OF EPILEPTIC ATTACKS.—Dr. Otto, *Berliner klin. Wochenschr.* publishes the results of his investigations as to the presence of albumen in the urine of epileptic patients. He analyzed the urine in thirty-one cases, and found one or more of the albumen reactions in twenty-two, fourteen times a precipitate and ten times turbidity. Hence,

he concludes that transitory albuminuria is to be considered one of the symptoms of the epileptic attack. Still, the frequent absence of this symptom and its short duration deprive it of any special practical importance.

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The following are among the articles recently published on the Pathology of the Nervous System and Mind and Pathological Anatomy.

WITKOWSKI, On the Melancholic Initial Stage of Insanity, *Berl. klin. Wochenschr.*, Dec. 11; BERNHARDT, Communication on Athetosis, *Deutsch. med. Wochenschr.*, Dec. 2; SARTISSON, Color Blindness and Railway Service, *St Petersburg med. Wochenschr.* Nov. 20—Dec. 2, 1876; MOOS, On the Connection between Diseases of the Auditory organs and those of the Fifth Nerve, *Virchow's Archiv.*, LXVIII, III, Nov. 13; A. MITCHELL, Contribution to the Statistics of Insanity, *Jour. of Mental Sci.*, Jan. 1877; D. HUCK TUKE, On the Prevalence of the causes of Insanity among the Ancients *Jour. of Ment. Sci.*, Oct. 1875; PEDDIE and BUCKNILL, Letters on the Relation of Drink and Insanity, *Ibid*; CARRE, Nervous Hemioptysis, *Arch. Gen. de Med.*, Jan. 1877; ALBERTONI, The Influence of the Brain on the Production of Epilepsy, *Archivio Italiano*, Nov. 1876; BOUCHUT, The Nature and Treatment of Tetany or Contraction of the Extremities in Infants, *Gaz. des Hopitaux*, Dec. 12; DU SAULLE, The Epileptics, *Ibid* (cont. art); HUGHLINGS JACKSON, On the Embolic Theory of Chorea *Brit. Med. Jour.* Dec. 23; MACKENZIE, Coincidence or Correlation? A Note on the Embolic Theory of Chorea, *Ibid.*; BERGER, On the Pathology of Rheumatic Facial Paralysis *Deutsche med. Wochenschr.* Dec. 9; BIANTE, General Paralysis as a predisposing cause of Fractures, *Ann. Med. Psych.* Nov. 1876; SIMON, The Imagination in Insanity, *Ibid.*

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## c.—THERAPEUTICS OF THE NERVOUS SYSTEM AND MIND.

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HYDROBROMIC ACID.—Dr. A. McLane Hamilton, *Phil. Med. Times*, Oct. 28, gives the following testimony on this agent.

"In appearance it is a straw-colored liquid, with an agreeable acid taste, and a slight odor of bromine. It combines very readily with many substances, and may be given with tinct. ferri. chlor., strychnine, etc. It prevents the headache caused by the iron, when given to persons who are anæmic (Fothergill). It dissolves a large amount of quinine; and Gubler found that the head-effects of that drug were not produced when this combination was employed."